SHORT REPORT

Popliteal Artery Entrapment Associated with Cannabis Arteritis

E. Ducasse,[1]* J. Chevalier,[1] D. Dasnoy,[1] F. Speziale,[2] P. Fiorani[2] and P. Puppinck[1]

[1]*Unit of Vascular Surgery, Catholic Institute of Lille, Lille, France; and* [2]*Department of Vascular Surgery, Clinic Umberto I, University "La Sapienza", Rome, Italy*

Objective. *To report popliteal artery entrapment in a patient with distal necrosis and cannabis-related arteritis, two rare or exceptional disorders never described in association. To conduct a targeted review and especially to seek information on the clinical presentation with characteristics specific to each disorder so as to hasten the diagnosis and choose appropriate management.*

Material and methods. *A 19-year-old man who presented with plantar claudication associated with necrosis in a toe underwent diagnostic arteriography and surgery for popliteal artery entrapment type III.*

Results. *Surgical clearance resolved the popliteal artery entrapment but left the clinical symptoms unchanged. Closer questioning disclosed a history of cannabis consumption and intravenous vasodilatory therapy was started. After the 21-day course of vasodilator agents the pain disappeared and the toe necrosis regressed. The patient stopped taking cannabis and had no signs of recurrence.*

Conclusion. *Whereas a popliteal artery entrapment, albeit a rare event, is well described and responds to standardized treatment, popliteal artery entrapment associated with cannabis-induced arteritis is an exceptional event that could confuse management. Because young people—the age group mainly at risk for popliteal artery entrapment—increasingly use cannabis, cannabis arteritis could become a more frequent event associated with other arterial disorders that may confuse the diagnosis and complicate management. Our experience in a young patient suggests that coexisting popliteal artery entrapment and distal necrosis in a young patient should raise a strong suspicion of an associated vascular disorder possibly related to cannabis consumption. Intravenous vasodilatation treatment is successful provided that cannabis use is discontinued.*

Key Words: Popliteal entrapment syndrome; Popliteal artery entrapment; Cannabis arteritis; Arteritis.

Introduction

Popliteal artery entrapment is a rare disorder that has a frequency of less than 0.5%[1] and affects a young male population.[2,3] The specific diagnosis is difficult and entails classifying the exact type of lesion. In most cases, surgical treatment provides a definitive diagnosis of the lesion and leads to the patient's recovery. Complicated popliteal entrapment remains an exceptional event and calls for a wide knowledge of the disease. Cannabis-related arteritis has been described in only 17 cases, all young patients. No published reports have described popliteal artery entrapment in a patient with cannabis-related arteritis. The case reported here and the targeted literature review is intended to clarify the clinical lesions and reported presentations, the diagnostic workup and the appropriate treatment for these two disorders, whether isolated or associated. The importance of this topic lies especially in the increasing consumption of cannabis in the occidental population.

Materials and Methods

We searched the literature for reviews, descriptions and classifications of popliteal artery entrapment; and for reported cases and reviews of arteritis induced by cannabis consumption. We used a combination of medical sub-headings and free text searches. Terms searched for included 'popliteal artery entrapment', 'popliteal entrapment', 'popliteal artery entrapment

*Corresponding author. Eric Ducasse, Department of Vascular Surgery, Hospital Pellegrin-Tripode, 33076 Bordeaux cedex, France.

syndrome', 'cannabis arteritis', 'drug arteritis' and 'arteritis'. The following databases were searched: MEDLINE from January 1966 to June 2003; EMBASE from January 1980 to June 2003 and the Database of Abstract of Reviews (DARE).

Case Report

A 19-year-old man with no history of vascular disease was admitted for investigation of distal necrosis in the third right toe. Over the previous three months, he had developed plantar claudication at 300 m. Clinical examination revealed a painful, white right foot, and a loss of the popliteal and foot pulses; pulses were present and normal on the left side. The patient was transferred to the vascular surgery unit for investigation and treatment. Arteriography, without postural manœuvres, disclosed no atheromatous lesions, but there was posterior popliteal artery compression in the right leg and unusually poor distal vascularization in both legs (Fig. 1). A magnetic resonance angiogram (MRA) obtained to distinguish between popliteal artery compression and entrapment from a parietal cyst found no cyst but failed to identify the type of compression or the causal agent. Laboratory blood chemical findings included a normal glucose and normal coagulation. The patient underwent surgery leading to a preliminary diagnosis of popliteal artery entrapment complicated by isolated distal necrosis. The surgical intervention confirmed, as initially suspected, a popliteal entrapment classified as type III.[4] The inter-condylar space was separated by a fibrous membrane that compressed the artery anteriorly and the vein posteriorly. The fibrous membrane was continuous with vertical fibres in the same place
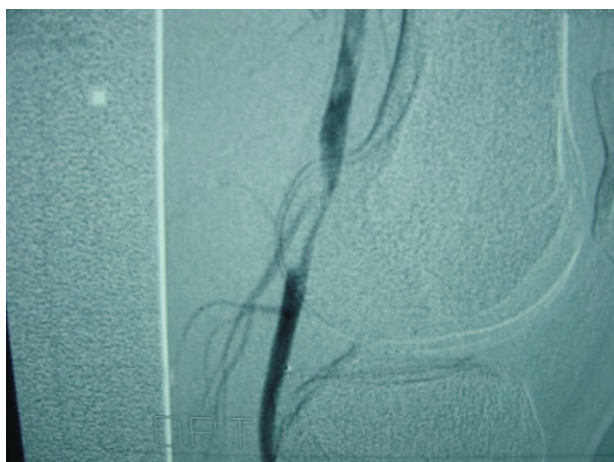
as the plantaris muscle tendon. This membrane was divided. During the immediate follow-up pain surprisingly failed to regress and the loss of distal pulses persisted despite the perfect result on the postoperative MRA follow-up scan (Fig. 2). On direct questioning, the patient admitted having consumed cannabis 10 times a day for 4 years. The history of cannabis abuse immediately suggested that the diagnosis of complicated popliteal artery entrapment with distal emboli might in fact be wrong; the signs might be due to a Buerger type complementary arteritis related to cannabis consumption. The patient attended the outpatient clinic for a 21-day course of intravenous vasodilators. Therapy induced a satisfactory clinical result, the leg pain disappeared and the toe necrosis regressed. A follow-up Doppler ultrasound examination at 1 month confirmed that though the distal arterial lesions persisted the hemodynamic status had enormously improved, and the upper arms were free of lesions. A peripheral MRA scan confirmed permanent occlusion of the three arteries on the right side of the leg and the peroneal artery on the left side (Fig. 3). A capillaroscopic examination gave normal findings

Fig. 1. Preoperative lateral angiography showing a tight stenosis of the right popliteal artery.

Fig. 2. Magnetic resonance angiogram obtained 8 days after surgical division of the fibrous band showing a complete regression of the popliteal artery stenosis.

Fig. 3. Magnetic resonance angiogram obtained three months after surgery and after medical treatment showing the right distal arterial lesions with thrombosis and poor distal run-off.

for the arms and legs, thus excluding Buerger's disease.

Discussion

This case of popliteal artery entrapment in a patient with distal necrosis and cannabis-related arteritis is instructive for several reasons. First, together with the literature review, it prompts a much needed overview of the symptoms, clinical presentations, exceptional complications and current treatment and follow-up of these two disorders. It also emphasises that popliteal artery entrapment in a young patient with non-specific symptoms should raise the suspicion of an associated cannabis-related lesion.

In accordance with reported data for popliteal entrapment, a rare disorder with a reported frequency of 0.5%,[1] the patient we describe here was a 19-year-old male. In a study conducted in a young male population Bouhoutsos et al.[1] estimated a frequency of 0.165%. In a post mortem study, Gibson reported a 3% frequency of popliteal artery entrapment.[5] The estimated male predominance ranges from 65 to 85%.[2,3] A

large review conducted in a mixed population nevertheless rejected masculine predominance.[6] Thus, this syndrome remains rare and predominantly affects a young population with no other arterial symptoms except a contralateral lesion, found in 20[2] to 66%[7] of cases. The mean age observed varies from 28[2,8] to 34.9.[9]

Our patient did not engage in competitive sports and the popliteal entrapment probably arose from an organic lesion. Classically, popliteal artery entrapment has two possible causes: organic or functional. The organic cause originates from an embryologic failure during the fourth week of gestation (20–22th day) when the future popliteal artery crosses the internal body of the gastrocnemius muscle. According to the arterial position and to the muscle insertion, the standard classification lists five types (type I to type V).[4] Although many have reported modifications or new classifications, as a guide to diagnosis, clinical management, surgical treatment and follow-up a complex classification is no substitute for a surgical exploration of the lesion and its anatomic relations.[2] In our patient, as in most reported cases, surgery classified a type III lesion of organic origin. Most lesions of functional origin arise from muscular compression secondary to hypertrophy after intensive physical training and exacerbated by the use of anabolic steroids.[10]

Clinically, the currently published reviews refer to 13–88 arteries analyzed.[3,6–9,11–13] The clinical symptoms—including distal claudication—depend on physical activity and complicated lesions may lead to permanent damage. Published reports describe complications in 0%[7,11–13] to 46%[3] (six arteries of 13 analyzed). Reported complications include a post-compression parietal reaction and post-stenotic aneurysmal dilatation leading to popliteal artery thrombosis and exceptionally, distal embolisms.[14] Complicated popliteal artery entrapment manifests itself clinically as critical leg ischæmia Fontaine stage III or IV, or acute severe leg ischæmia. No published reports have described entrapment of the popliteal artery alone causing distal claudication and focal necrosis, the signs we observed in the young cannabis abuser described here.

Considering that the case of popliteal artery entrapment we report concerns a 19-year-old man, the most instructive finding in our review of popliteal artery entrapment is the absence of complications in patients younger than 40 years. In the review conducted by Levien et al. including the largest number of complications (18 arteries), none of these complications involved patients younger than 38 years.[9] In the review conducted by Turnipseed et al.,

patients with complicated forms underwent surgery at an older mean age than patients with simple presentations (43.6 years versus 24.5 years).[6] As our case underlines, popliteal artery entrapment diagnosed in a young patient with a distal lesion should also raise a suspicion of a second lesion, most probably caused by arteritis.

In our patient, clinical examination showed a permanent loss of distal pulses, and Doppler and angiographic exploration confirmed a tight popliteal artery stenosis unexpectedly associated with bilateral distal arterial thrombosis. In current clinical practice, popliteal artery entrapment is investigated using clinical examination, Doppler and angiographic exploration, looking for the loss of arterial pulse, echo signal, or opacification on postural movements such as knee hyper-extension or maximum active plantar flexion. Our patient did not undergo these manœuvres during angiography because a tight stenosis was immediately seen. The only specific finding on exploration was a type I tortuous popliteal artery.[4] These diagnostic investigations yield a large number of false positive results—as they do for the thoraco-brachial outlet syndrome—ranging from 30% in a young population to 50% in an athletic population.[15] Our experience shows that arterial exploration should be used also for detecting other arterial lesions (for example, arteritis in a young population) or associated complications (including aneurysmal dilatation). Although popliteal artery entrapment is also diagnosed with CT scan or MRI these imaging procedures contribute little to the specific diagnosis, the search for associated lesions and even less to treatment.[8,16]

At surgery for popliteal artery entrapment in the young man described here, we decompressed the popliteal artery by resecting the plantaris muscle and tendon. Arterial decompression by an appropriate muscular or tendon resection is the classic surgical treatment for the non-complicated forms of popliteal artery entrapment. In the functional forms of entrapment, these techniques could be traumatizing, especially for athletes.[2] Our literature review shows that the complicated forms are more difficult to treat. In some cases, arterial wall degeneration responds to endarterectomy and venous patching alongside fibrous or muscular decompression, but as for other complications—post-stenotic dilatation and thrombosis—popliteal artery replacement using a vein graft produces the best results with fewer complications.[3,11]

In our patient successful surgical clearance of the popliteal artery entrapment unexpectedly left the patient's clinical symptoms (plantar pain and necrosis) unchanged. Only after surgery and after questioning the patient did we diagnose cannabis-related arteritis, an exceptional disorder. Since the first case report in 1960 by Sterne *et al.*,[17] only 17 cases of cannabis-related arteritis have been published, principally in France (Tables 1 and 2).[18–23] The vasoconstrictor effect of drugs of abuse has been well known since the 1960s. Cocaine, synthetic amphetamine and LSD are potent vasoconstrictors; cannabis is less potent. The vasoconstrictor action of cannabis (delta 8 and 9 Tetrahydrocannabinol—THC) has been well demonstrated, especially associated with arsenic.[24,25] Even though no visceral lesion have been described in cannabis use, unlike other drug-induced lesions,[26,27] anatomically, cannabis-induced lesions match those described for Buerger's disease but develop faster and after lower cannabis consumption than the equivalent tobacco consumption required to produce the changes of Buerger's disease.

In line with the findings in our patient with cannabis-related arteritis, our literature review showed that cannabis abuse usually leads to distal arterial lesions with thrombosis and a collateral development of helical arteries. Claudication in the lower limbs involves the plantar area. As it did in our patient, necrosis is present initially or develops rapidly.[18–20] Like Buerger's disease, lesions of upper arms are less frequent but in 25% of the cases are the principal lesion.[20] Whereas our patient had no ultrasound evidence of upper arm arterial lesions, asymptomatic upper-arm arterial lesions are associated with lower limb arterial lesions in two-thirds of the cases.[28] Proximal lesions have been described in four cases with two bilateral amputations during follow-up.[20,22] Despite a similar clinical presentation, the major difference from Buerger's disease is the absence of intraparietal inflammatory cells.[17]

Our patient's persisting clinical symptoms responded well to postoperative medical treatment with intravenous prostacyclin. This finding agrees with the reported use of this vasodilator to resolve distal symptoms.[18–23] In the absence of inflammatory mechanisms conservative management depends almost entirely on stopping cannabis consumption and possibly on secondary sympathectomy to increase the collateral flow.[20] Two case reports describe recurrent arteritis in patients who continued taking cannabis.[20,21]

Another instructive point emerging from our literature review is the homogeneous clinical presentation (Table 1). In 90% of the cases (16/18 including our patient) distal necrosis with punctiform features developed within weeks. In at least 50% of the cases, patients reported a recent onset of distal claudication. This clinical presentation matches that of auto-

Table 1. Descriptions of lesions reported in the literature for cannabis arteritis (17 patients + our case report)

Patient	Age	Sex	Clinical lesion			Arterial examination	
			Limb	Distal necrosis	Clinic symptoms	Distal pulses	Arteriographic findings
1 (17)	18	M	Lower	I	Claudication	Absent bilaterally	Occlusion 1/3 > TA and TP
2 (20)	20	M	Upper	I and II (R)		Absent cubital bilaterally	Palm occlusion
3 (20)	30	M	Upper	III (R) and V (L)		Absent bilaterally	Digital occlusion R and L
4 (20)	29	M	Lower	No	Acute ischemia (R)	Absent bilaterally	femoropopliteal occlusion
5 (20)	40	M	Lower	I (L)	Claudication	Absent (L)	TA and TP occlusion, ilio-femoral vasoconstriction
6 (20)	38	M	Lower	I and V (L)	–	Absent (L)	TA and TP occlusion
7 (20)	22	M	Lower	I (R)	Claudication	Absent bilaterally	Femoropopliteal occlusion
8 (20)	20	M	Lower	I and II (R)	Claudication	Absent (R)	TA and TP
9 (20)	35	M	Lower	I (L)	Claudication	Absent bilaterally	–
10 (20)	30	M	Upper	III (L)	–	Absent cubital bilaterally	–
11 (20)	38	M	Lower	I (R)	Claudication	Absent (R)	TA et TP occlusion
12 (21)	21	M	Lower	Yes (type ?)	–	Absent	Distal occlusion
13 (21)	31	M	Upper	Yes (type ?)	–	Absent	Distal occlusion
14 (21)	20	M	Lower	Yes (type ?)	–	Absent	Proximal occlusion
15 (21)	23	M	Lower	Yes (type ?)	–	Absent	Distal occlusion
16 (23)	19	M	Lower	III (R)	Claudication	Absent (R)	TA and TP occlusion
17 (22)	30	F	Lower	No	Claudication	Absent	Proximal lesions
Our case	19	M	Lower	III (R)	Claudication	Absent	Distal occlusion

R, right; L, left; arteries: TA, tibial anterior artery; TP, tibial posterior artery; –, data not reported.

immune arteritis, with absent distal pulses, and permanent angiographic obliteration of the distal arterial segments. The permanent angiographic features of this symptomatic arterial disorder have never been described in a patient with uncomplicated popliteal artery entrapment. The elevated serum homocysteine level is of no help in diagnosing cannabis-related arteritis. Our patient had a 4-year history of daily cannabis consumption (10 spliffs a day). Most patients described in our review had on average an 8-year history of moderate cannabis usage. Many cases refer to the associated use of tobacco, and only two to consumption of other drugs of abuse.

In conclusion, popliteal artery entrapment associated with distal complications in a young patient should raise the suspicion of an associated disorder. The more widespread use of recreational drugs, including cannabis, in the Western population may increase the number of drug-induced arterial disorders requiring management in clinical practice.

Table 2. Description of drug abuse and follow-up reported in the literature for cannabis arteritis (17 patients + our case report)

Patients	Intoxication			I.P.T.H.L.	Stop cannabis	Follow-up
	Tobacco	Cannabis	Others			
1 (17)	10/day (1 year)	1/day (1 year)	No	No	No	–
2 (20)	20/day (6 years)	2/day (3 years)	No	No	Yes	Good
3 (20)	20/day (12 years)	5/day (1 year)	Ectasy	Yes	–	–
4 (20)	20/day (10 years)	– (–)	No	No	–	Bilateral amputation
5 (20)	– (–)	– (20 years)	No	Yes	–	–
6 (20)	20/day (20 years)	– (23 years)	Heroin	Yes	–	–
7 (20)	20/day (5 years)	2/week (5 years)	No	–	Yes	Good
8 (20)	20/day (4 years)	4/day (4 years)	No	No	No	Recurrence-death
9 (20)	20/day (10 years)	1/day (10 years)	No	No	Yes	Good
10 (20)	20/day (15 years)	5/day (12 years)	No	–	Yes	Good
11 (20)	20/day (10 years)	– (10 years)	–	No	Yes	Good
12 (21)	20/day (4 years)	– (–)	No	–	Yes	Good
13 (21)	20/day (4 years)	– (–)	No	–	Yes	Good
14 (21)	20/day (5 years)	– (–)	No	–	No	Bilateral amputation
15 (21)	20/day (3 years)	– (–)	No	–	No	Recurrence-good
16 (23)	20/day (5 years)	10/day (4 years)	No	No	Yes	Good
17 (22)	20/day (10 years)	1/day (16 years)	No	No	No	Good
Our case	5/day (5 years)	10/day (4 years)	No	No	Yes	Good

I.P.T.H.L, increased plasma total homocysteine level (>15 mmol/l); –, data not reported.

References

1 BOUHOUTSOS J, DASKALAKIS E. Muscular abnormalities affecting the popliteal vessels. *Br J Surg* 1981; **68**: 501–506.

2 STAGER A, CLEMENT D. Popliteal artery entrapment syndrome. *Sports Med* 1999; **28**: 61–70.

3 SCHURMANN G, MATTFELDT T, HOFFMAN W, HOHENBERGER P, ALLENBERG JR. The popliteal entrapment syndrome: presentation, morphology and surgical treatment of 13 cases. *Eur J Vasc Surg* 1990; **4**: 223–231.

4 INSUA JA, YOUNG JR, HUMPHRIES AW. Popliteal artery entrapment syndrome. *Arch Surg* 1970; **101**: 771–775.

5 GIBSON MH, MILLS JG, JOHNSON JE, DOWNS MD. Popliteal entrapment syndrome. *Ann Surg* 1977; **185**: 341–348.

6 TURNIPSEED W. Popliteal entrapment syndrome. *J Vasc Surg* 2002; **35**: 910–915.

7 COLLINS PS, MCDONALD PT, LIM RC. Popliteal artery entrapment: an evolving problem. *J Vasc Surg* 1989; **10**: 484–489.

8 ROSSET E, HARTUNG O, BRUNET C, BRUNET C, ROCHE PH, MAGNAN PE *et al.* Popliteal artery entrapment syndrome. Anatomic and embryologic bases, diagnostic and therapeutic considerations following a series of 15 cases with a review of the literature. *Surg Radiol Anat* 1995; **17**: 161–169.

9 LEVIEN LJ, VELLER MG. Popliteal artery entrapment syndrome: more common than previously recognized. *J Vasc Surg* 1999; **30**: 587–598.

10 LEPORI M, PERREN A, GALLINO A. The popliteal-artery entrapment syndrome in a patient using anabolic steroids. *N Engl J Med* 2002; **346**: 1254–1255.

11 HOELTING T, SCHUERMANN G, ALLENBERG JR. Entrapment of the popliteal artery and its surgical management in a 20 years period. *Br J Surg* 1997; **84**: 338–341.

12 LAMBERT AW, WILKINS DC. Popliteal artery entrapment syndrome: collaborative experience of the joint vascular research group. *Br J Surg* 1998; **85**: 1367–1368.

13 DESHPANDE A, DENTON M. Functional popliteal entrapment syndrome. *Aust N Z J Surg* 1998; **68**: 660–663.

14 BERGER HA. Insidious symptomatology and misleading physical findings in popliteal artery entrapment syndrome. A case report. *J Cardiovasc Surg* 1999; **40**: 861–863.

15 RIGNAULT D, PAILLER JL, LUNEL F. The functional popliteal entrapment syndrome. *Int Angio* 1985; **4**: 341–343.

16 JANS L, VERSTRAETE KL, HUYSSE WC. Popliteal artery entrapment by an aberrant muscular slip. *JBR-BRT* 2002; **85**: 132–133.

17 STERNE J, DUCASTING C. Cannabis India arteritis. *Arch Mal Coeur* 1960; **53**: 143–146.

18 DISDIER P, SWIADER L, JOUGLARD J, PIQUET PH, CHRISTIDES C, MOULIN G *et al.* Cannabis-induced arteritis vs. Leo Buerger disease. Nosologic discussion a propos of two new cases. *Presse Med* 1999; **28**: 71–74.

19 CONSTANT J, DUBIEZ P, BASTE JC, BARCAT D, PARROT F, CONRI C. Cannabis arteriopathy. *Presse Med* 1999; **28**: 2154.

20 DISDIER P, GRANEL B, SERRATRICE J, CONSTANT J, MICHON PASTUREL U, HACHULLA E *et al.* Cannabis arteritis revisited. Ten new case reports. *Angiology* 2001; **52**: 1–5.

21 CAZALETS C, LAURAT E, CADOR B, ROLLAND Y, JEGO P, GROSBOIS B. Cannabis arteritis: four new cases. *Rev Med Interne* 2003; **24**: 127–130.

22 SCHNEIDER F, ABOUCHELI-BAUDOT N, TASSART M, BOUDGHENE F, GOUNY P. Cannabis and tobacco: cofactors favoring juvenile obliteration arteriopathy. *J Mal Vasc* 2000; **25**: 388–389.

23 SCHNEIDER HJ, JHA S, BURNAND KG. Progressive arteritis associated with cannabis use. *Eur J Vasc Endovasc Surg* 1999; **18**: 366–367.

24 ADAMS MD, EARNHARDT JT, DEWEY WL, HARRIS LS. Vasoconstrictor action of delta 8 and 9 Tetrahydrocannabinol in the rat. *J Pharmacol Exp Ther* 1976; **196**: 649–656.

25 NOËL B. Regarding 'cannabis arteritis revisited: ten new case reports'. *Angiology* 2001; **52**: 505–506.

26 HALPERN M, CITRON BP. Necrotizing angiitis associated with drug abuse. *Am J Roentgenol Radium Ther Nucl Med* 1971; **111**: 663–671.

27 MÖKEL M, KAMPF D, LOBECK H, FREI U. Severe panarteritis associated with drug abuse. *Intensive Care Med* 1999; **25**: 113–115.

28 MICHON PASTUREL U, QUEREL V, LE BERRE V, HACHULA E, HATRON PY, DEVULDER B. Arteritis-related cannabis: an entity to discover. *J Mal Vasc (suppl B)* 1999; **24**: 157–159.